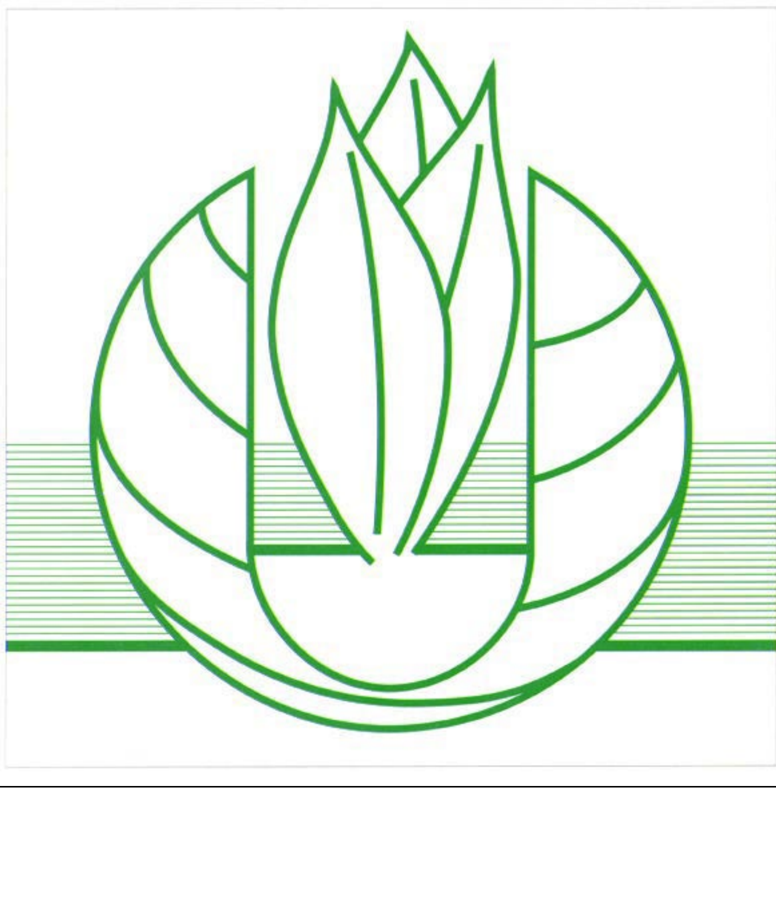
FAO/IPGRI Technical Guidelines for the Safe Movement of Germplasm. No.14

Small Grain Temperate Cereals

edited by **M. Diekmann and C.A.J. Putter**



in collaboration with the
International Center
for Agricultural Research
in the Dry Areas

PREVIOUSLY PUBLISHED TECHNICAL GUIDELINES FOR THE SAFE MOVEMENT OF GERMPLASM

Cocoa	1989
Edible Aroids	1989
Musa	1989
Sweet Potato	1989
Yam	1989
Legumes,	1990
Cassava	1991
Citrus	1991
Grapevine	1991
Vanilla	1991
Coconut	1993
Sugarcane	1993
Small fruits *(Ragaria, Ribes, Rubus, Vaccinium)*	1994

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INTRODUCTION

Collecting, conservation and utilization of plant genetic resources and their global distribution are essential components of international crop improvement programmes.

Inevitably, the movement of germplasm involves a risk of accidentally introducing plant quarantine pests[1] along with the host plant material; in particular, pathogens which may accompany symptomless host material, such as viruses, pose a special risk. In order to minimize this risk, effective testing (indexing) procedures are required to ensure that distributed material is free of pests that are of quarantine concern.

The ever-increasing volume of germplasm exchanged internationally, coupled with recent rapid advances in biotechnology, has created a pressing need for crop-specific overviews of the existing knowledge in all disciplines relating to the phytosanitary safety of germplasm transfer. This has prompted FAO and IPGRI to launch a collaborative programme for the safe and expeditious movement of germplasm, reflecting the complementarity of their mandates with regard to the safe movement of germplasm. FAO, as the depository of the International Plant Protection Convention of 1951, has a long-standing mandate to assist its member governments to strengthen their Plant Quarantine Services, while IPGRI's mandate - *inter alia* - is to further the collecting, conservation and use of the genetic diversity of useful plants for the benefit of people throughout the world.

The aim of the joint FAO/IPGRI programme is to generate a series of crop-specific technical guidelines providing relevant information on disease indexing and other procedures that will help to ensure phytosanitary safety when germplasm is moved internationally.

The technical guidelines are produced through meetings of panels of crop experts selected in consultation with the relevant specialized institutions and research centres. The experts contribute to the elaboration of the guidelines in their private capacities and do not represent the organizations for whom they work. The guidelines are intended as advice to institutions involved in germplasm exchange and FAO, IPGRI and the contributing experts cannot be held responsible for any failures resulting from the application of the present guidelines. By their nature, they reflect the consensus of the crop specialists who attended the meeting, based on the best scientific knowledge available at the time of the meeting. The experts who have contributed to this document are listed after this introduction.

The technical guidelines are written in a short, concise style, in order to keep the volume of the document to a minimum and to facilitate updating. Suggestions for further reading are given at the end, along with the references cited in the text (mostly for geographical distribution, media and other specific information only). The guidelines are divided into two parts. The first part makes general recommendations on how best

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[1] The word 'pest' is used in this document as it is defined in the International Plant Protection Convention. It encompasses all harmful biotic agents ranging from viroids to weeds.

to move germplasm of the crop concerned and mentions available intermediate quarantine facilities when relevant. The second part covers the important pests and diseases of quarantine concern. The information given on a particular pest or disease does not pretend to be exhaustive but concentrates on those aspects that are most relevant to quarantine.

In the present guidelines small grain temperate cereals are covered, i.e. bread wheat *(Triticum aestivum* L.), durum wheat *(Triticum durum* Desf., [syn. *T. turgidum* L. var. *durum* Desf.]), barley *(Hordeum vulgare* L.), rye *(Secale cereale* L.), oat *(Avena sativa* L.) and triticale (x Triticosecale Wittmack). In the text, reference is made to common names only. A mention of 'wheat' signifies bread and durum wheat.

The present guidelines were developed at a meeting held in Aleppo, Syria from 20 to 22 March, 1994. The meeting was hosted by the International Center for Agricultural Research in the Dry Areas (ICARDA).

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General Recommendations

- In this publication the term 'germplasm' refers to breeding material as well as genebank material (including wild relatives and progenitors)[2].
- All cereal germplasm should be maintained free of seed-associated pests specified in these guidelines or in any countries' regulations.
- Only accessions certified to be free of such pests should be distributed.
- For genebank material, accessions which are not yet in a pathogen-tested state should be handled according to the same procedures as described for new introductions.
- In recipient countries, seedlots should be established and maintained for one generation under conditions of appropriate isolation (temporal and/ or spatial), with periodic inspection, testing, roguing and chemical protection if necessary
- Seedlots should be tested for seed-associated pests and certified by the appropriate regulatory agency before distribution.

Technical Recommendations

- Seeds should be harvested at a time optimal for the crop and care should be taken to ensure effective drying.
- Seeds should be collected from plants free from specified seed-associated pests.
- Seed samples should be cleaned to eliminate all soil, plant debris, contaminant pests and seeds of noxious weeds.
- Seeds should be given appropriate pesticide treatments, unless otherwise specified by the recipient country.
- Seedlots suspected to contain insects should be fumigated with an appropriate fumigant. Alternatively small samples might be subjected to a temperature below -15°C for at least one week.
- Parcels containing seeds should be unpacked in a closed (insect-proof) area, and packing material should be incinerated or autoclaved.

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[2] Commercial seedlots should continue to be subject to current regulatory procedures.

Movement of germplasm

1. Introduction of germplasm

Introduction of new germplasm entries should satisfy regulatory requirements of the recipient country.

Each new introduction should be grown in appropriate isolation. Plants should be observed periodically. Plants suspected to be affected with specified seed-associated pests should be destroyed.

Seeds should be sown in appropriate isolation with chemical protection if necessary.

Plants should be observed periodically. Plants affected by specified seed-associated pests should be removed and destroyed.

2. International distribution of germplasm

Movement of germplasm should comply with the regulatory requirements of the recipient country. Germplasm shipments should always be accompanied by a phytosanitary certificate.

In addition to the phytosanitary certificate a 'germplasm health statement' indicating which tests have been performed to assess the health status of the material may accompany the germplasm.

If newly introduced germplasm is moved without multiplication, the shipment should be accompanied by a re-forwarding phytosanitary certificate and a copy of the original phytosanitary certificate.

Definitions of Terms as Used in this Publication

Seeds
The fertilized ripened ovules or fruits (caryopses) of cereals.

Internally seedborne
Any structure (unit) of pathogen inside the seed, e.g. in the embryo *(Ustilago nuda)*, endosperm *(Xanthomonas campestris)* or pericarp *(Pyrenophora graminea).*

Externally seedborne
Any structure (unit) of pathogen contaminating the surface of pericarp or hull (lemma, palea) of some barley (e.g. *Tilletia tritici).*

Admixtures
Any structure (unit) of pathogen or pest mixed with the seeds, e.g. propagules, infected plant debris, etc.

Seed-transmitted vs. seedborne
While ‘seedborne’ implies only the presence of a pathogen in, on, or with the seed, ‘seed-transmitted’ includes the pathogen’s passage from seeds to seedlings and plants.

Cosmopolitan
This expression is used to describe the distribution of pathogens which are reported to occur in all continents, and in many countries of these continents.

Seedborne Pests in Small Grain Cereals

Note: All pathogens listed are potentially seed-transmitted; only pathogens in categories A and B are included in these guidelines.

Pathogen	A: internally seedborne	B: externally seedborne	C: see admixtures
Virus			
Barley stripe mosaic virus	x		
Bacteria			
Clavibacter tritici	x	x	
Erwinia rhapontici	x	x	
Pseudomonas fuscovaginae	x	x	
Pseudomonas syringae pv. *atrofaciens*	x	x	
Pseudomonas syringae pv. *striafaciens*	x	x	
Pseudomonas syringae pv. *syringae*	x	x	
Xanthomonas campestris pv. *translucens*	x	x	
Fungi			
Alternaria triticina	x	x	
Bipolaris sorokiniana (Helminthosporium sativum)	x	x	
Claviceps purpurea	o[1]		x
Cochliobolus victoriae (oat)	x		
Fusarium graminearum			x
and other species causing head scab	x		
Leptosphaeria nodorum (Septoria nodorum)	x		
Mycosphaerella graminicola (Septoria tritici)			
Phaeosphaeria avenaria (Septoria avenae)			
Pyrenophora graminea (Helminthosporium gramineum)	x		
Pyrenophora teres (Helminthosporium teres)	x		
Pyrenophora tritici-repentis			
(Helminthosporium tritici-repentis)	x		
Pyricularia oryzae	x	x	
Sclerophthora macrospora	x	x	
Tilletia caries and *T. foetida*	o	x	x
Tilletia controversa	o	x	x
Tilletia indica	x	x	x
Urocystis agropyri		x	
Urocystis occulta		x	
Ustilago avenae		x	
Ustilago hordei		x	
Ustilago nuda	x		
Ustilago tritici	x		
Nematodes			
Anguina agrostis (barley)	o		
Anguina tritici	o		
Aphelenchoides fragariae (oat)	o		x
Heterodera avenae			x
Heterodera latipons			x
Heterodera zeae (oat)			x

[1] o = whole seed is transformed into a perennation and infectious structure of the pathogen as a result of infection. According to ISTA rules (1993) this structure is no longer considered a seed.

For general references see p. 56.

Descriptions of Pests

Viral diseases

Barley stripe mosaic

Cause
Barley stripe mosaic virus is a hordeivirus with straight, tubular particles about 22 nm in diameter and of two to four lengths (100 to 150 nm), depending on the strain. It is readily transmitted in sap (Atabekov and Novikov 1989).

Significance
High incidence and severity in Montana and North Dakota barley crops in the 1950s (Carroll 1980).

Symptoms
Stripe mosaic on cereal hosts (Fig. 1). Symptoms may vary from very mild stripe mosaic to lethal necrosis depending on the host genotype and environmental conditions (Fig. 2). Symptoms may be confused with barley stripe disease *(Pyrenophora graminea).*

Hosts
- natural: The virus has a narrow host range. Natural hosts known are barley, wheat and wild oat (McKinney and Greeley 1965).
- experimental: 240 members of the Gramineae, nine members of the Chenopodiaceae and one member each of the Solanaceae, Amaranthaceae and Primulaceae have been infected.

Fig. 1. Chlorotic and necrotic stripe symptoms of barley stripe mosaic (BSV) on barley. (Dr T.W. Carroll, Montana State University, Bozeman, MT)

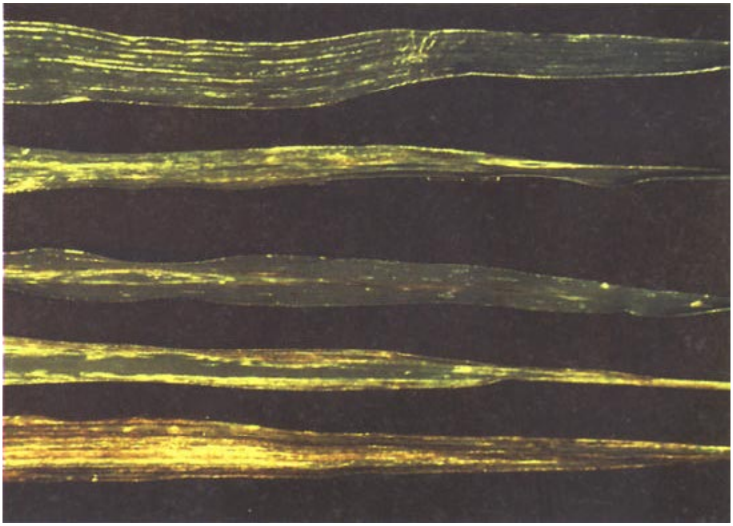

Fig. 2. Variability of chlorotic and necrotic stripe symptoms caused by BSMV infection. (Dr T. W. Carroll, Montana State University, Bozeman, MT; previously published in Plant Dis. 64: 136-140 (1980); printed with permission from American Phytopathological Society)

Geographical distribution
Cosmopolitan.

Biology and transmission
No insect vector has been reported to transmit the virus. Seed transmission rate in barley can reach up to 90%; even 100% (Atabekov and Novikov 1989) has been reported. The virus is also pollen-borne and infects the pollinated plants (Gold *et al.* 1954; Gardner 1967). The main route of natural spread of the virus in the field seems to be by plant-to-plant contact.

Detection
Mechanical inoculation of *Chenopodium amaranticolor* and *C. quinoa* produces local lesions, which are useful for detection. Serology is very useful for virus detection, as the virus is highly immunogenic. Several tests can be employed such as gel diffusion (Carroll *et al.* 1979), ELISA (Lister *et al.* 1981), and more recently tissue-blot immunoassay (Makkouk and Kumari, unpublished).

Treatment
No direct treatment; ELISA testing and elimination of infected lots.

For further reading, see p. 56.

Bacterial diseases

1. Bacterial leaf blight

Cause
Pseudomonas syringae pv. *syringae* van Hall

Significance
Foliage destruction may exceed 50%, but yield losses due to bacterial leaf blight have not been thoroughly assessed (Wiese 1987).

Symptoms
Bacterial leaf blight develops on the uppermost leaves after plants reach the boot stage. Initially, small, water-soaked lesions appear and may expand and coalesce into irregular streaks or blotches (Fig. 3) within 2 to 3 days under cool, wet conditions. Lesions turn from greyish-green to tan or white as tissues become necrotic. Ears and glumes may occasionally be infected, resulting in tan to brown necrotic spots with distinct margins.

Hosts
Pseudomonas syringae pv. *syringae* has a multiplicity of hosts, including many dicots. Some strains infect wheat, barley, oat, rye and triticale.

Geographical distribution
Cosmopolitan.

Biology and transmission
The role of seed transmission in establishing the disease is unknown.

Detection
Routine microbiological procedures for isolation and culture are recommended.

Treatment
Hot water treatment of seed at 53°C for 30 min, and seed treatment with phytobactiomycin, quinolate, falisan and carboxin are reported to be effective (Koleva 1981).

For further reading, see p. 57.

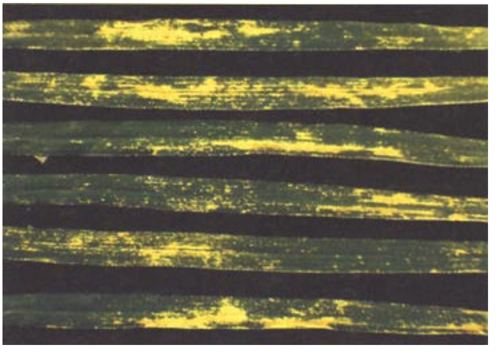

Fig. 3 Lesions of bacterial leaf blight caused by *Pseudomonas syringae* pv. *syringae*. (Dr J. D. Otta, Sioux Falls, SD; previously published in Compendium of Wheat Diseases; printed with permission from American Phytopathological Society)

2. Bacterial sheath rot

Cause
Pseudomonas fuscovaginae Miyajima, Tanii & Akita.

Significance
This pathogen causes an important disease of rice, but its significance in wheat is unknown.

Symptoms
Irregular, angular, blackish brown lesions bordered by a purple-black water-soaked area (Fig. 4) (similar to those of bacterial sheath brown rot of rice). Lesions are necrotic and may be 10 to 20 cm in length.

Hosts
Wheat, rice.

Geographical distribution
On wheat reported from Mexico (Duveiller and Maraite 1990).

Biology and transmission
No information for wheat, but seedborne in rice.

Detection
Standard procedures apply (Schaad 1988); no specific procedures reported.

Treatment
There is no known treatment to eradicate the pathogen.

For further reading, see p. 57.

Fig. 4 Lesions of bacterial sheath rot caused by *Pseudomonas fuscovaginae* (Dr E. Duveiller, CIMMYT, El Batan)

3. Basal glume rot (basal glume blotch, spikelet rot)

Cause
Pseudomonas syringae pv. *atrofaciens* (McCulloch) Young, Dye & Wilkie [syn. *P. atrofaciens* (McCulloch) F.L. Stevens]; *Phytomonas atrofaciens* (McCulloch) Bergey *et al.*

Significance
Yield losses due to poor seedfill have been reported (Wiese 1987). The status of the pathogen in the Ukraine is attracting considerable research attention (Pasichnik and Koroleva 1991).

Symptoms
Water-soaked, dark green to brown lesions tend to be inconspicuous on unripe wheat heads. Darkened or streaked tissues often are confined to the base of glumes where they are more conspicuous on the inner surface. Diseased kernels have a faint brown to black discolouration on their base (Fig. 5). In advanced stages, kernels are shrunken and stained brown-black at their embryo end.

Hosts
Wheat, barley, oat, rye.

Geographical distribution
Australia, Bulgaria, Canada, former Czechoslovakia, Morocco, New Zealand, Romania, Russia (Siberia), South Africa, Ukraine, USA, Zimbabwe (CMI 1982). Reported as a new record on durum wheat in Syria (Mamluk *et al.* 1990).

Biology and transmission
Seedborne.

Detection
Standard procedures apply (Schaad 1988); no specific procedures reported.

Treatment
There is no known treatment to eradicate the pathogen.

For further reading, see p. 57.

Fig. 5. Symptoms caused by basal glume rot (*Pseudomonas syringae* pv. *atrofaciens*) on wheat seeds. (Dr J. von Kietzell, Institut für Pflanzenpathologie und Pflanzenschutz der Universität, Göttingen)

4. Black chaff (bacterial leaf streak, bacterial streak)

Cause

Xanthomonas campestris pv. *translucens* (Jones, Johnson & Reddy) Dye [syn. *Bacterium translucens* var. *undulosum* Smith, Jones & Reddy]. Related pathovars include *X.c.* pv. *undulosa* (Smith, Jones & Reddy) Dye, *X.c.* pv. *secalis* (Reddy, Godkin & Johnson) Dye, and *X.c.* pv. *cerealis* (Hagborg) Dye.

Significance

Yield losses are difficult to quantify precisely, but estimates range from negligible to more than 40% (Shane 1985; Forster and Schaad 1988; Mehta 1990).

Symptoms

Black blotches on glumes of wheat (Fig. 6) (hence the name black chaff); water-soaked lesions (streaks or spots) on awns, peduncle and leaves (Fig. 7) leading to tan or translucent necrotic lesions (except purple to black lesions on peduncle, occasionally with a yellow centre). The alternating bands of healthy and infected tissue on awns produce a 'barber pole' appearance which is a useful diagnostic feature in the field. Dried bacterial exudate on leaf surfaces may be present and appears as small, thin, semi-transparent flakes (referred to as 'shellac') or small, yellow, crystalline droplets (Fig. 8). Symptoms on wheat glumes are similar to a non-infectious condition known as false black chaff or melanism caused by expression of the SR-2 gene. Symptoms on glumes may also resemble those caused by *Leptosphaeria nodorum* in regions where both diseases occur. Hence, diagnosis must include isolation of the pathogen.

Hosts

Wheat, barley rye, triticale.

Geographical distribution

Widely distributed, occurring in many of the principal cereal-producing countries of the world (Duveiller 1989; CAB IMI 1993). Some confusion exists due to problems with identification and taxonomy of closely related pathovars.

Fig. 6. Black streaks and blotches on glumes are symptomatic of black chaff, but may be confused with other diseases. (Dr R.L. Forster, University of Idaho, Kimberly, ID)

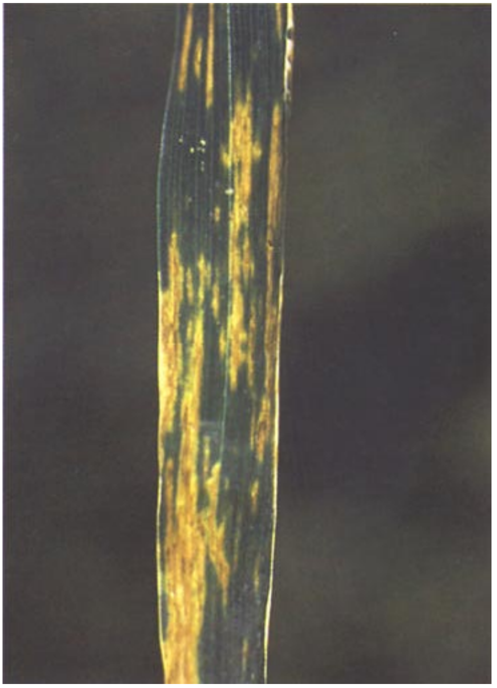

Fig. 7. Brown necrotic lesions, which are often surrounded by a lime-green halo, are typical symptoms of black chaff (bacterial leaf streak, bacterial streak) on wheat leaves. (Dr R.L. Forster, University of Idaho, Kimberly, ID)

Biology and transmission
Seedborne externally and internally Survives on crop debris, gramineous weeds (Wallin 1946; Boosalis 1952) and on healthy leaves.

Detection
The pathogen is easily isolated from infected leaf and stem tissue on several media including nutrient glucose agar, YDC agar and semi-selective XTS agar.

Treatment
Seed soaking in hot, acidified cupric acetate will eradicate the pathogen (Forster and Schaad 1988; Duveiller 1989), but the treatment can be phytotoxic. Heat treatment (72°C for 5 to 7 days) has been found effective (Fourest et *al.* 1990).

For further reading, see p. 57.

Fig. 8. Small yellow droplets of bacterial ooze are characteristic of black chaff. (Dr M. Diekmann, IPGRI, Rome)

5. Halo blight of oat

Cause
Pseudomonas syringae pv. *coronafaciens* (Elliott) Young *et al.* [syn. *Pseudomonas coronafaciens* (Elliott) Stevens]. Schaad and Cunfer (1979) proposed that P. *striafaciens* and several other pathovars and subspecies of *P. coronafaciens* were all synonyms of *P. coronafaciens.*

Significance
Halo blight is common in most oat-growing regions of the world, but generally causes little yield reduction unless severe infection occurs prior to emergence of the panicle (Dickson 1956).

Symptoms
Small, tan coloured spots with greasy looking margins (Fig. 9) develop initially and may elongate, darken and develop a prominent translucent halo. In severe infections, leaves yellow and dry

Hosts
- natural: oat.
- experimental: wheat and barley

Geographical distribution
Cosmopolitan. For detailed list, see Bradbury (1986). The disease was reported for the first time in Brazil in 1986 (Reis *et al.* 1986).

Biology and transmission
Seedborne.

Detection
Standard procedures apply (Schaad 1988); no specific procedures reported.

Treatment
There is no known treatment to eradicate the pathogen.

For further reading, see p. 58.

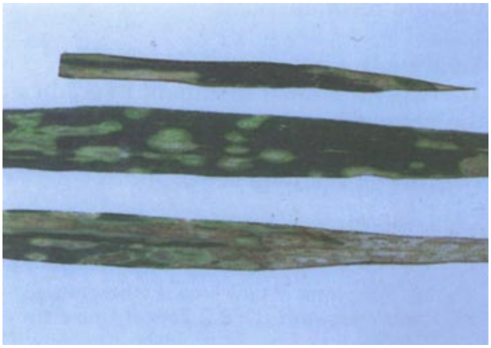

Fig. 9. Lesions of halo blight of oat caused by *Pseudomonas syringae* pv. *coronafaciens.* (Dr N.W. Schaad, USDA-ARS, Frederick, MD)

6. Pink seed

Cause
Erwinia rhapontici (Millard) Burkholder [syn. *Erwinia carotovora* pv. *rhapontici* (Millard) Dye].

Significance
Pink seed causes cosmetic effects and is considered inconsequential (Wiese 1987).

Symptoms
Seeds harboring the pathogen/parasite have a light pink appearance (Fig. 10) due to the presence of a diffusible pigment produced by the bacterium in the testa. These symptoms are similar to the colouration of seeds treated with fungicides containing dyes and to seeds from plants infected by *Fusarium* species, the causal agents of scab or head blight. Wheat spikes artificially inoculated with the pathogen develop a maroon colour in the spikelet tissue surrounding the inoculation point.

Hosts
Wheat, rhubarb *(Rheum rhaponticum)*, hyacinth *(Hyacinthus orientalis)*, pea *(Pisum sativum)*.

Geographical distribution
USA (Idaho, North Dakota), Canada, England, France, Russia, Ukraine. (Roberts 1974; Sellwood and Lelliott 1978; CMI 1981; McMullen *et al.* 1984; Forster and Bradbury 1990; Huang *et al.* 1990).

Biology and transmission
Seedborne.

Detection
Routine microbiological procedures (seed plating and streaking) for isolation and culture are recommended.

Treatment
There is no known treatment to eradicate the pathogen.

For further reading, see p. 58.

Fig. 10. Symptoms of pink seed of wheat caused by *Erwinia rhapontici*. (Dr R.L. Forster, University of Idaho, Kimberly, ID)

7. Spike blight (yellow ear rot, yellow slime, or tundu) of wheat

Cause

Clavibacter tritici (Carlson & Vidaver) Davis, Gillaspie, Vidaver & Harris [syn. *Corynebacterium michiganense* pv. *tritici* (Hutchinson) Dye & Kemp; *Corynebacterium tritici (ex* Hutchinson) Carlson & Vidaver); *Corynebacterium rathayi* (E.F. Smith) Dowson].

Significance

Spike blight is dependent on the nematode vector *Anguina tritici.* The disease caused heavy losses in earlier years, but has become extinct or rare in most parts of western Europe, North America, Australasia and the former USSR due to efficient control of the nematode.

Symptoms

Production of bright yellow bacterial slime or gum on the leaf surfaces of young plants and on the aborted leaves and ears in contact with them at the boot stage. The culm is always distorted when the ears have spike blight symptoms.

Hosts

- natural: bread wheat, also the grasses *Alopecurus monspeliensis*, *Lolium temulentum*, and *Phalaris minor*.

- experimental: by inoculation, using the nematode vector, susceptibility was shown for durum wheat, *Triticum dicoccum*, and *T. pyramidale.*

Geographical distribution

Australia, China (Hopeh, Kweichow), Cyprus, Egypt, Ethiopia, India, Iran (CMI 1978).

Biology and transmission

Seedborne via the nematode *Anguina tritici*, which is the only vector.

Detection

See Anguina tritici.

Treatment

Control of *Anguina tritici* prevents the disease.

For further reading, see p. 58.

Fungal diseases

1. Alternaria leaf blight of wheat

Cause

Alternaria triticina Prasada & Prabhu. Conidia of *A. triticina* vary from 7 to 30 µm in width and 15 to 90 µm in length, are dark, ellipsoid to conical (Fig. 11), tapering to a beak and arise singly or in short chains (2 to 4) from dark conidiophores.

Significance

Epidemics have been reported to occur in India in the 1960s (Prabhu and Prasada 1970).

Symptoms

The pathogen may infect all foliar parts. Discoloured oval lesions appear on lower leaves. As the plant matures the disease progresses upwards and lesions enlarge and coalesce to irregular, dark blotches, often with chlorotic margins (Fig. 12). Under humid conditions, dark powdery masses of conidia may be seen on lesions. Seeds severely infected with the pathogen are discoloured and shriveled. However, low infection levels do not result in obvious symptoms.

Fig. 11. Spores of *Alternaria triticina.* (CIMMYT, El Batan)

Fig. 12. Alternaria leaf blight on wheat. (CIMMYT, El Batan)

Hosts
Wheat, barley, triticale.

Geographical distribution
South Asia, West Asia, North Africa (Agarwal *et al.* 1993), Nigeria, Mexico (Waller 1981) and Italy (Frisullo 1982).

Biology and transmission
A. triticina survives in host crop residues and seed. The leaves touching the soil are infected first. Moderate temperatures around 20 to 25°C, light rainfall and/or heavy dew favour infection.

Detection
The fungus is not easily differentiated from the many *Alternaria* species commonly associated with seed. The detection of *A. triticina* in seed may be facilitated by plating disinfested seeds on the medium recommended by Agarwal *et al.* (1993).

Treatment
Many fungicides control external but not internal inoculum. However, treating seed with iprodione and thiram is claimed to provide effective control (Raut *et al.* 1983). An alternative method is soaking seed in water at 52 to 54°C for 10 min (Prabhu and Prasada 1970).

For further reading, see p. 58.

2. Bunt diseases

2.1. Common bunt of wheat

Cause
Two morphologically different fungi: (1) *Tilletia laevis* Kühn [syn. *T. foetida* (Wallr.) Liro., *T. foetens* (Berk. & Curt.) Schroet.] and (2) *T. tritici* (Bjerk.) Winter [syn. *T. caries* (DC.) Tul.]. The two pathogens differ mostly in their spore wall structure: *T. laevis* has a smooth, *T. tritici* a reticulated surface (Fig. 13).

Significance
Common bunt reduces yield and grain quality. The disease is ranked second after rusts in world-wide importance (Hoffmann 1982; Wiese 1987). It is of major importance where seed treatment is not practised (Hoffmann 1982; Mamluk and Zahour 1993). In Syria, 50% of the surveyed fields between 1984 and 1988 showed common bunt infection with many of the fields expressing a high incidence (up to 60%) of infected plants (Mamluk *et al.* 1990; Mamluk and Zahour 1993).

Symptoms
The pathogens affect only the inflorescence, replacing kernels by a bunt ball containing a black, powdery mass of teliospores (Fig. 14). Bunted plants are sometimes shorter than healthy ones; the glumes may be spread apart because of the larger bunt ball (Fig. 15). The pericarp remains intact, but ruptures easily during threshing. A fish-like odour (trimethylamine) is characteristic for the infected kernels. Infected kernels are sometimes confused with kernels infected by *Anguina tritici;* the latter being smaller and not breaking easily. In rare cases, partial infection occurs, which may lead to confusion with *Tilletia indica.*

Hosts

- natural: wheat including *T. dicoccum,* and species of the genera *Aegilops, Agropyron, Lolium, Arrhenatherum, Bromus, Dactylis* (Hoffmann and Schmutterer 1983), and *Elymus* and *Hordeum* spp. (Wiese 1978).

- experimental: *T. boeoticum* and *T dicoccoides* (Mamluk and van Slageren 1993) as well as *Aegilops kotschyi* and *Ae. tauschii* (ICARDA 1993).

Geographical distribution
Cosmopolitan.

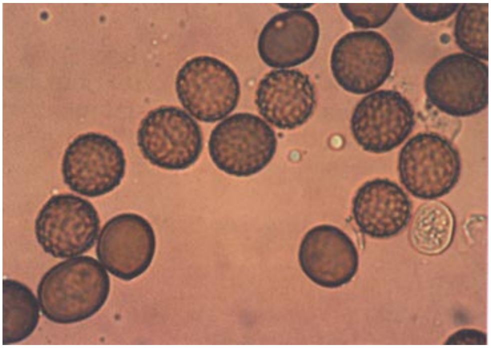

Fig. 13. Teliospores of *Tilletia laevis* (smooth surface, e.g. bottom left) and *Tilletia tritici* (reticulated surface, e.g. centre). (Dr O.F. Mamluk, ICARDA, Aleppo)

Biology and transmission

Primary source of inoculum are the externally seedborne teliospores. Seed may be naturally contaminated with as many as 230,000 spores/seed. Teliospores may also be soilborne, and can survive for at least 2 years in dry soil. Teliospore germination depends largely on soil temperature and moisture. Infectious hyphae penetrate the coleoptile before seedlings emerge. The coleoptile is susceptible only as long as it is not split open.

Detection

Direct examination of seed for bunt balls; centrifuge wash test and microscopic investigation. Confusion in spore appearance may occur between those of *T. tritici* and of *T. controversa* (Stockwell 1990).

Treatment

Chemical seed treatment, such as carboxin and carboxin in combination with thiram. However, soilborne inoculum may not be adequately controlled (Line 1993).

For further reading, see p. 59.

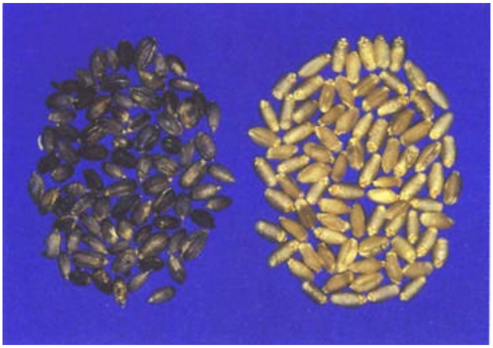

Fig. 14. Right: healthy seeds;
Left: bunt balls containing spores of *Tilletia tritici.*
(Dr. O.F. Mamluk, ICARDA, Aleppo)

Fig. 15. Left: Spike of durum wheat showing symptoms of common bunt; right: healthy spike. (Dr O.F. Mamluk, ICARDA, Aleppo)

2.2. Dwarf bunt

Cause

Tilletia controversa Kühn; sometimes the incorrect spelling *T. contraversa* is found in the literature. Teliospores show large polygonal reticulation with spiny projections and are surrounded by a gelatinous sheath (Fig. 16).

Significance

Important where no appropriate seed treatment is used. From Bavaria, Germany yield losses of 30% were reported (Smith *et al.* 1992). Prevalent in areas where temperature just above freezing prevails for several months, and under snow cover. In West Asia and North Africa occurring only at altitudes above 1100 m. Bunted spikes result only from high levels of seed infestation (more than 20,000 teliospores per seed).

Symptoms

Affected plants are stunted to various extents. Tillering may be increased. Kernel symptoms resemble those of common bunt.

Hosts

In addition to wheat, rye and barley a number of grasses (*Aegilops* spp., *Aguopyron* spp., *Alopecurus myosuroides, Arrhenatherum elatius, Bromus* spp. *Dactylis glomerata, Elymus* spp., *Festuca* spp., *Koeleria cristata, Lolium* spp., *Poa* spp. (Smith *et al.* 1992).

Geographical Distribution

Afghanistan, Albania, Algeria, Argentina, Australia, Austria, Bulgaria, Canada, former Czechoslovakia, Denmark, France, Germany, Greece, Hungary, Iran, Iraq, Italy, Japan, Libya, Morocco, New Zealand, Poland, Romania, Spain, Sweden, Switzerland, Syria, Tunisia, Turkey, Uruguay, USA, former USSR, former Yugoslavia (CAB IMI 1992).

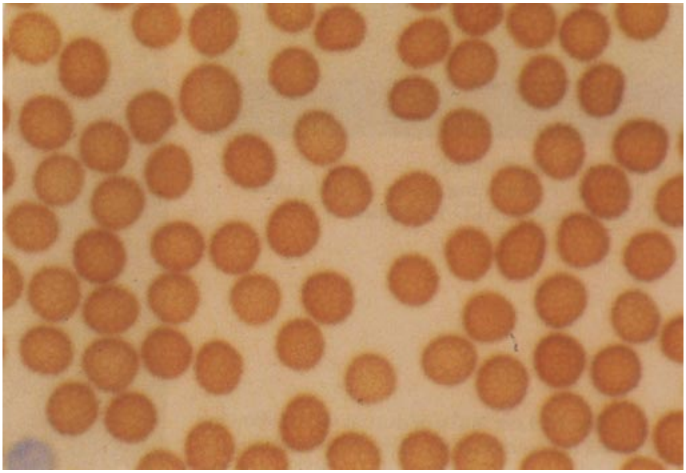

Fig. 16. Teliospores of *Tilletia controversa,* surrounded by a gelatinous sheath. (Dr A. El-Ahmed, ICARDA, Aleppo)

Biology and transmission

It is soilborne and externally seedborne, but the majority of dwarf bunt infections result from soil infestation. Teliospores can remain viable for 3 to 10 years in soil or on seeds.

Detection

Direct inspection for infected seeds and centrifuge wash test for spore contamination. Fluorescence microscopy may help to distinguish the spores from those of *T. tritici:* the reticulated wall layer of teliospore fluoresces yellow-orange (Stockwell and Trione 1986).

Treatment

Seed treatment with systemic fungicides such as Difenoconazole (Sitton *et al.* 1993) or NaOCl (0.13 M at maximum 55°C, Chastain 1991). Soilborne inoculum is generally difficult to control.

For further reading, see p. 59.

2.3. Karnal bunt of wheat

Cause
Tilletia indica Mitra [syn. *Neovossia indica* (Mitra) Mundkur] (Fig. 17).

Significance
Disease distribution is limited by climate and primarily associated with spring wheat crops grown in dry, irrigated areas. Disease occurs rarely in dryland crops and never in true winter wheat crops. Durum wheats are rarely affected.

Symptoms
Disease symptoms are not easily observed in the field but obvious in threshed seeds. Infection begins at the embryo end of the seed and, depending upon the environmental conditions during grain maturation, proceeds along the crease of the seed (Fig. 18). Partial infection is typical. Infected seeds usually retain the pericarp; the disease is confined to the endosperm of the seed which is converted to a mass of dry black teliospores. These teliospores often emit a fishy odor, as observed in other bunt diseases.

Hosts

- natural: wheat, triticale.
- experimental: *Aegilops triuncialis, A. cylindrica, A. bicornis, A. comosa, A. searsii, A. tauschii, A. triaristata, Bromus ciliatus, Lolium multiflorum, L. perenne, Triticum monococcum* and *T. timopheevii* (Royer *et al.* 1986).

Geographical distribution
Afghanistan, India, Iraq, Mexico (CMI 1989), Nepal (Singh *et al.* 1989), Pakistan (CMI 1989), Brazil (Da Luz *et al.* 1993).

Biology and transmission
Seedborne and soilborne teliospores are the main source of inoculum.

Germinating seeds or seedlings are not systemically invaded. Rather, teliospores germinate on or very close to the surface of the soil producing a promycelium bearing filiform primary haploid

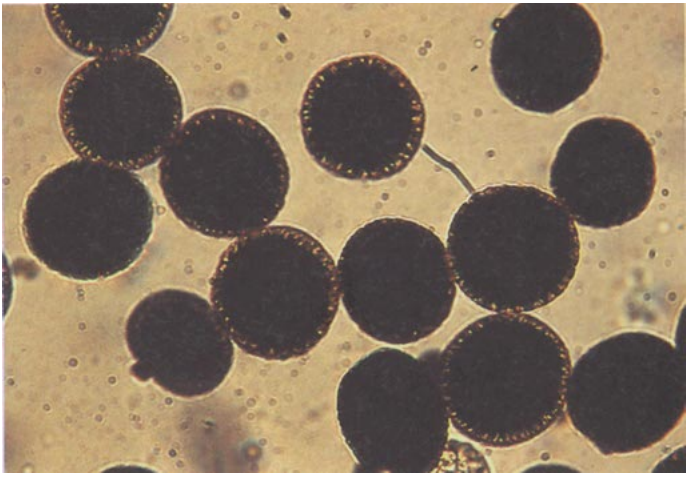

Fig. 17. Teliospores of *Tilletia indica.* (CIMMYT, El Batan)

Fig. 18. Wheat seeds showing symptoms of Karnal bunt. (CIMMYT, El Batan)

sporidia. These sporidia, or the haploid secondary allantoid sporidia produced subsequently, are carried to spikes by wind currents and/or splashing rain droplets. Spikelets are locally infected at the embryo end by diploid mycelia produced by the fusion of two mating types. Depending on the weather and the stage of seed maturity, the pathogen may become partially systemic in rachides and rachillae and produce additional infections. Infection is favoured by rainfall, cloud cover, heavy dew formation and cool temperatures (15 to 20°C) about the time of flowering.

Detection

The pathogen may be detected as infected seed and/or seed-borne teliospores. Infected seed may be seen with the naked eye or with the aid of a low-power magnifying glass. Detection may be improved by augmenting the contrast between the dark sori and the lighter uninfected areas by soaking seeds for 24 hours in a solution of 0.1% NaOH. Common black point is often confused with Karnal bunt. Always confirm identification by the presence of teliospores. Teliospores are characteristically large, more or less spherical, and dark (immature ones are lighter in colour). The mean diameter, including a sheath, ranges between 33 and 40 µm.

Teliospores may be easily detected in seed washates. Wash 25 to 50 g seeds in 100 ml water (+ 1 drop Tween 20) for 30 min; filter the wash water through a Whatman #l paper filter in a Büchner funnel. Use a small laboratory pump to quickly extract the liquid. Wet the filter paper with 1 to 3% KOH (to expose the sheath surrounding the teliospore) and observe under a stereomicroscope at a magnification of 25-50. Mark dark, shiny, spherical bodies of about 35 µm with small pieces of coloured plastic. Always confirm identification under a compound microscope. Conidia of *Epicoccum* spp. are often confused with teliospores of *T. indica;* however, they are reticulated and only about 25 µm in size.

Treatment

Pentachloronitrobenzene (PCNB) or chlorothalonil have given the best results as seed treatments, although all fungicides tested thus far are fungistatic to some degree. Further, teliospores protected by the seed pericarp and/or spore masses inside intact sori may be protected from the chemical. However, teliospores on seed surfaces are eliminated by disinfesting seeds in a 1% solution of sodium hypochlorite (+ Tween 20) with agitation for 3 min.

For further reading, see p. 60.

3. Downy mildew

Cause
Sclerophthora macrospora (Sacc.) Thirum., C. Shaw & Narasimhan [syn. *Sclerospora macrospora* Sacc.].

Significance
The disease occurs occasionally in wet areas. However, the destructive potential of the disease under favourable conditions warrants careful monitoring of its presence and spread.

Symptoms
Infected wheat plants are erect, yellowish green, dwarfed to some extent, and they tiller excessively. The leaves are thickened, remain erect, and develop in a close whorl around the culm because of reduced internodal elongation and the stiff thickened conditions of the leaf blade. Many of the infected tillers turn brown and die. Inflorescences may be malformed, and seeds shriveled. The large brown oospores in the mesophyll tissue between the veins of the leaf blade and sheath constitute the important diagnostic symptom.

Hosts
Wheat, barley oat, triticale, maize and *Sorghum vulgaris* (Miles and Epps 1942; Ullstrup 1952; Troutman and Matejka 1972; Fuentes 1974). Annual and perennial grasses as listed by Semeniuk (1976) and Semeniuk and Mankin (1964 1966).

Geographical distribution
Cosmopolitan.

Biology and transmission
Primary source for new infections is oospores, which remain viable for years in soil, dead plant tissue and seeds of wheat and oat. Germinating oospores produce sporangiophores which germinate to release motile zoospores. Under free moisture conditions these can infect plants systemically in any stage of development.

Detection
Microscopical inspection of seeds or seed washings for presence of oospores, measuring about 60 µm in diameter.

Treatment
No additional specific treatment can be recommended.

For further reading, see p. 60.

4. Head scab (Fusarium head blight)

Cause

The principal pathogens are listed in Table 1.

Significance

Wheat quality as well as yield can be substantially reduced by scab infection. Two toxins, zearalenone and deoxynivalenol (vomitoxin or DON), are produced in 'scabby' grain. The environment in which the cereal is grown determines which species of *Fusarium* causes head scab. *Fusarium graminearum* predominates in areas where the climate is warm and where maize and rice are also grown. *Fusarium nivale* and *Fusarium avenaceum* cause the disease in cool areas, the latter fungus occurring particularly in association with pasture grasses and legumes. *Fusarium culmorum* predominates in areas intermediate between these extremes, i.e. in moderate to cool areas (Bergstrom 1993).

Symptoms

Head scab causes premature bleaching or death of individual spikelets, parts of the ear or the entire ear (Fig. 19). Infected spikelets are often sterile. Infection of the rachis can cause the whole head to become bleached. In severely diseased heads and under humid weather, a dark brown discolouration may extend from the diseased spikelets onto the peduncle. White mycelium and pink to orange sporodochia of the fungus can often be seen at the edges of the glumes or at the base of the spikelet. As the head matures, small, black perithecia may become visible on the glumes. Seeds from diseased heads may show a pink discolouration and be small and shrivelled. Known as 'tombstones', diseased grain can be removed by grading or winnowing with a fan. When diseased seed is planted, seedling blight may develop and seedlings die prior to or after emergence. Less severe infection may result in disease of the coleoptile, subcrown internode, leaf sheaths and primary roots. This may result in the development of 'foot rot', crown and root rot of adult plants.

Fig. 19. Head scab caused by *Fusarium graminearum.* (Dr R.G. Rees, Queensland Wheat Research Institute, Toowoomba, Qd)

Table 1. Pathogens causing head scab.

TELEOMORPH	ANAMORPH	SPORE MORPHOLOGY	CULTURE CHARACTERISICS
Principal pathogens			
Gibberella zeae (Schwein.) Petch	*Fusarium graminearum* Schwabe Group 2	macroconidia 2.5-5 x 35-62 µm, 5 to 7 septate, sickle shaped, with foot cell	gray, pink and buff with a carmine colour in the agar
	Fusarium culmorum (W.G. Smith) Sacc.	macroconidia 4-7 x 25-50 µm, 3 to 5 septate, with foot cell	yellow-brown, discolours agar media to red-brown
Other pathogens			
Monographella nivalis (Schaffnit) E. Müller	*Fusarium nivale* (Fr.) Ces. ex Berl. & Vogl. [syn. *Microdochium nivale* (Fries) Samuels & Hallett]	macroconidia 2.8-4 x 16-25 µm, 1 to 3 septate, without an evident heel	white to peach-coloured colonies
Gibberella avenacea Cook	*Fusarium avenaceum* (Fr.) Sacc.	macroconidia 3.5-4 x 40-80 µm, 4 to 7 septate with elongated apical cell and prominent foot cell; microconidia 3.0-4.4 x 8-50 µm, 1 to 3 septate	rose-red, fringed with white becoming yellow-brown at the centre of the culture
	Fusarium crookwellense Burgess, Nelson & Toussoun	macroconidia 1.2-6.8 x 33.7-53.7 µm; majority of spores intermediate between *F. graminearum* and *F. culmorum;* foot cells very distinct	pale orange, becoming reddish brown to dark brown with age

Hosts

- natural: barley, rye, wheat, triticale, maize, numerous grasses (Sprague 1950).

- experimental: *Triticum dicoccum* Schübler, *Triticum polonicum* L., *Triticum spelta* L., *Triticum turgidum* L. (Christensen *et al.* 1929).

Geographical distribution
Cosmopolitan.

Biology and transmission
Most of the epidemiological studies have been with *F. graminearum.* For this fungus, host debris on or near the soil surface is the principal site of survival. Scab does not occur from seedborne inoculum, but from either macroconidia or ascospores. It may also develop from chlamydospores or hyphal fragments. Infection occurs principally between the anthesis and soft dough stages. It is favoured by continuous wetness of the spikelet (minimum 24 h) and temperatures of 20 to 30°C.

Detection
Freezing blotter method. Fine, white mycelium can be seen on the seed surface with shiny sporodochia of macroconidia. Sporodochia range in colour from pale to dull orange in *F. graminearum,* from dull orange to whitish pink in *F. culmorum* and from bright orange to red in *F. avenaceum.*

Treatment
Seed treatment with triadimefon, propiconazole, benomyl or methyl 2-benzimidazole (MBC) can reduce scab (Jacobsen 1977; Martin and Johnston 1982; Boyacioglu *et al.* 1992).

For further reading, see p. 61.

5. 'Helminthosporium' diseases

5.1. Barley stripe

Cause
Teleomorph: *Pyrenophora graminea* (Died.) Ito & Kuribay, anamorph: *Drechslera graminea* (Rab. ex Schlecht.) Shoem. [syn. *Helminthosporium gramineum* (Rab. ex Schlecht.)]. The conidia are straight with rounded ends, subhyaline to yellow-brown, with up to seven transverse septa, and measure 11 to 24 x 30 to 110 µm. Perithecia are rare in nature.

Significance
Potentially important in areas where seed treatment is not practised (Mathre 1982). Yield reductions may reach up to 70% under epidemic conditions (Pant and Bisht 1983).

Symptoms
One or more long chlorotic stripes start from the basal portion of the leaf blade. Parallel to the leaf ribs, stripes extend gradually toward the leaf tip and become necrotic (Fig. 20). Stripes usually coalesce and cause the tissue to split; eventually the entire leaf dies. Affected plants are usually stunted; several tillers fail to develop heads, or heads fail to emerge. Most spikelets of the emerged heads are blighted and turn brown. Seeds are shrivelled and may show a brown discolouration.

Hosts
Barley.

Geographical distribution
Cosmopolitan

Biology and transmission
The pathogen is strictly seedborne. It survives as mycelium in the hull, pericarp and seed coat, but not in the embryo. Sprinkler irrigation, rain and wind favour conidia production on infected leaves and their

Fig. 20. Chlorotic and necrotic stripes caused by *Pyrenophora graminea.* (Dr C. J. Langerak, CPRO-DLO, Wageningen)

dispersal. Seed infection occurs during flowering and until the hard-dough stage. Infection of seedlings from seed is highest at soil temperature below 12ºC and at intermediate soil moisture levels during seed germination. The ratio infected seeds/infected seedlings is approximately 1:0.4 (Porta-Puglia *et al.* 1986).

Detection
Blotter/freezing blotter method, growing-on method.

Treatment
Imazalil seed treatment (Johnston *et al.* 1982; Tekauz *et al.* 1985).

For further reading, see p. 61.

5.2. Net blotch

Cause

Teleomorph: *Pyrenophora teres* (Died.) Drechs., anamorph: *Drechslera teres* (Sacc.) Shoem. Conidiophores are olive to brown, single or in groups of two or three. The conidia are subhyaline to olive-green, straight and cylindric, with rounded apical cells, 1 to 11 septate, and measure 15 to 23 x 30 to 175 µm. Pseudothecia are 1 to 2 mm in diameter, and covered with dark setae. Ascospores are 18 to 28 x 43 to 61 µm, light brown, ellipsoidal, with three transverse septa and one or two longitudinal septa in the median cells.

Significance

Important disease in areas where barley is cultivated continuously. Losses of 10 to 40% are common but losses of nearly 100% occur in susceptible barley varieties (Mathre 1982). Grain quality may also be reduced.

Symptoms

Two apparent forms of symptoms are produced on leaves and leaf sheaths. *Pyrenophora teres* f. *teres* incites symptoms characterized by typical net blotch lesions that have dark brown striations extending longitudinally and transversely within the lesion to form a netlike pattern (Fig. 21). *Pyrenophora teres* f. *maculata* produces spot symptoms, namely dark brown circular or elliptical lesions (without netting), surrounded by a chlorotic zone. The spot form could be confused with the spot blotch symptoms caused by *Cochliobolus sativus.*

Hosts

- natural: barley and *Bromus diandrus.*
- experimental: wheat, *Aegilops* spp., *Agropyron* spp., *Avena* spp., *Bromus* spp., *Lolium* spp., and several other gramineous species (Shipton *et al.* 1973).

Geographical distribution

Cosmopolitan.

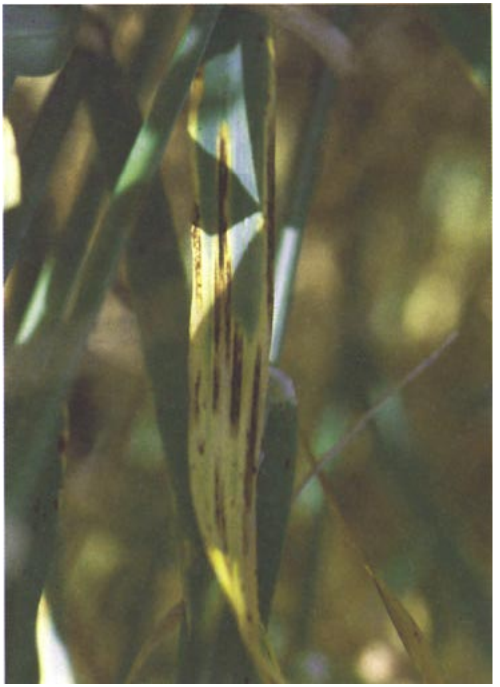

Fig. 21. Net blotch on barley. (Dr A. El-Ahmed, ICARDA, Aleppo)

Biology and transmission

The fungus overwinters as dormant mycelium in the seed or as pseudothecia in infected host residue. Seedborne mycelium probably plays a role in the introduction into previously pathogen-free areas, whereas conidia produced from lesions contribute to secondary infection. Whereas seedling infection is greatest at temperatures of 10 to 15°C, temperatures of 15 to 25°C are conducive to conidia production and secondary infection. The disease is common in temperate regions, and in areas of high humidity and rainfall, although epidemics have occurred in dry areas (Mathre 1982).

Detection

Blotter or freezing blotter method, growing-on method.

Treatment

Imazalil (Sheridan and Grbavac 1985).

For further reading, see p. 62.

5.3. Spot blotch

Cause
Teleomorph: *Cochliobolus sativus* (Ito & Kurib.) Drechs. ex Dast., anamorph: *Bipolaris sorokiniana* (Sacc.) Shoem. [syn. *Helminthosporium sativum* Pam. King & Backe, *Helminthosporium sorokinianum* Sacc. ex Sorok.]. Conidiophores are brown, mostly single and bear 1 to 6 conidia at the end. Conidia are dark brown, mostly straight or slightly curved, ellipsoid and thick-walled (Fig. 22). They are 6 to 9 septate and measure 17 to 24 x 68 to 99 µm.

Significance
Significant in warm areas.

Symptoms
Brown, roundish lesions, sometimes with yellow halos, on leaves and leaf sheaths (Figs. 23, 24). Sporulation of older lesions accounts for their olive-brown appearance.

Hosts
Wheat, barley, oat, rye, triticale and many grasses.

Geographical distribution
Cosmopolitan.

Biology and transmission
Seedborne as well as soilborne, also overwinters in plant residues.

Detection
Blotter test.

Treatment
Seed treatment, e.g. with Baytan.

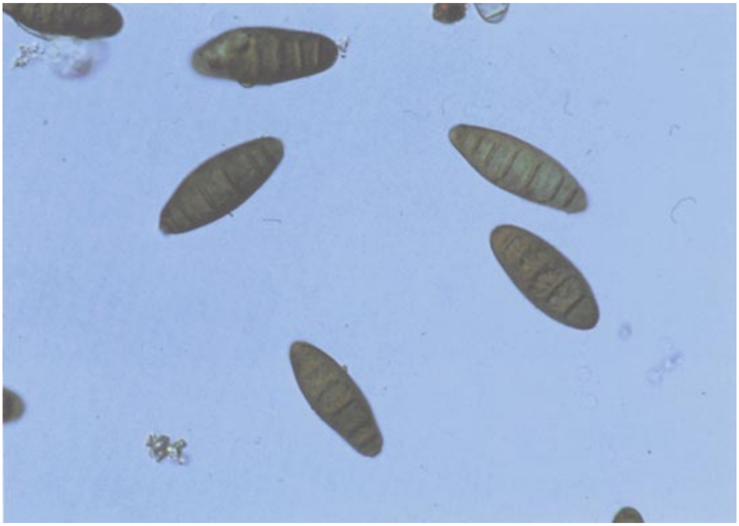

Fig. 22. Conidia of *Bipolaris sorokiniana.* (Dr G.B. Wildermuth, Queensland Wheat Research Institute, Toowoomba, Qd)

Fig. 23. Symptoms of leaf spot caused by *Bipolaris sorokiniana* on wheat: brown lesions with yellow halo. (Dr R.G. Rees, Queensland Wheat Research Institute, Toowoomba, Qd)

Fig. 24. Symptoms of stem (left) and head (right) infection caused by *Bipolaris sorokiniana* on wheat. (Dr R.G. Rees, Queensland Wheat Research Institute, Toowoomba, Qd)

5.4. Tan spot, yellow leaf spot

Cause

Teleomorph: *Pyrenophora tritici-repentis* Drechs. [syn. *P. trichostoma* (Fr.) Fuckel], anamorph: *Drechslera tritici-repentis* (Died.) Shoem. [syn. *Helminthosporium tritici-repentis* Died.]. Conidiophores are olive-brown, erect, and measure 7 to 8 x 100 to 300 µm. Conidia are subhyaline, cylindrical, with 4 to 7 septa, and measure 12 to 21 x 45 to 200 µm. Ascospores (18 to 28 x 45 to 70 µm) are oval to globose, brown, with three transverse septa and one or two longitudinal septa in the median cells.

Significance

It is significant in areas where wheat stubble is retained. Yield losses may reach approximately 50% (Rees *et al.* 1982).

Symptoms

Tan-brown flecks, expanding into lens-shaped lesions up to 12 mm long (Fig. 25). Often surrounded by a yellow border and with a dark brown spot in the centre. Lesions may coalesce, causing large necrotic areas of the older leaves.

Hosts

- natural: wheat, rye, *Bromus* spp., *Agropyron* spp. (Wiese 1987); barley, (Mathre 1982).

- experimental: many grass species (Krupinsky 1992b).

Geographical distribution

Cosmopolitan.

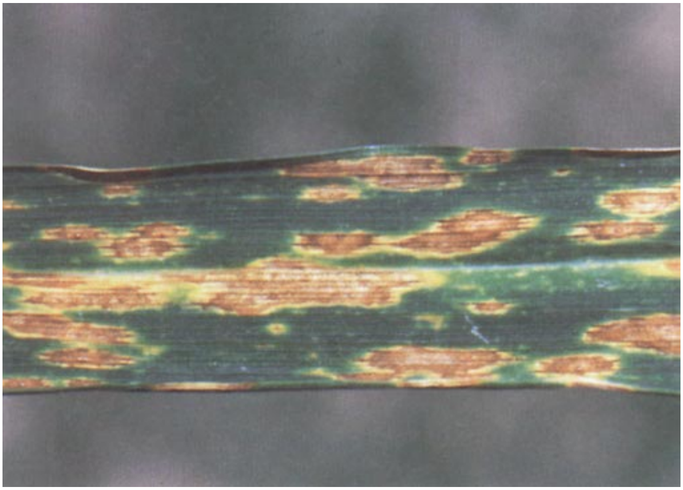

Fig. 25. Lens-shaped lesions of tan spot on wheat. (Dr R.G. Rees, Queensland Wheat Research Institute, Toowoomba, Qd)

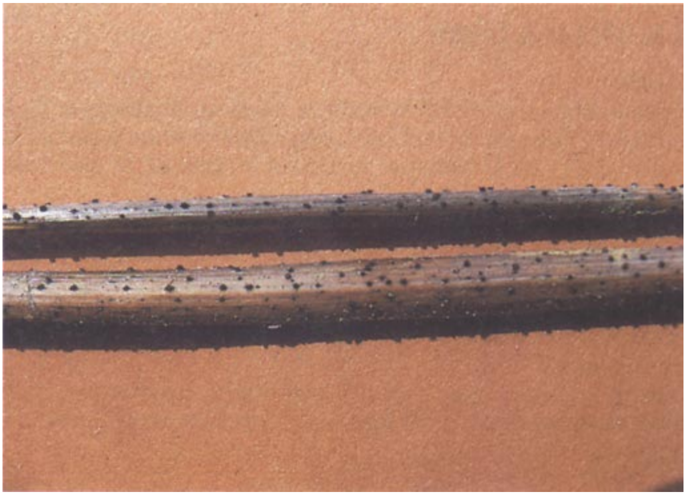

Fig. 26. Dark pseudothecia of *Pyrenophora tritici-repentis* on wheat straw. (Dr A. El-Ahmed, ICARDA, Aleppo)

Biology and transmission

The pathogen survives saprophytically as mycelia and pseudothecia in or on infested host debris under wet conditions (Fig. 26). Ascospores, conidia, and mycelia serve as primary inoculum, whereas seedborne inoculum appears insignificant. Conidia developed on lesions serve as secondary inoculum; their production and release are favoured by rain or dew.

Detection

No specific methods known.

Treatment

None available.

For further reading, see p. 62.

5.5. Victoria blight

Cause
Teleomorph: *Cochliobolus victoriae* Nelson, anamorph: *Drechslera victoriae* (Meehan & Murphy) Subram. & Jain [syn. *Helminthosporium victoriae* Meehan & Murphy]. Conidia morphology of *D. victoriae* is similar to that of *D. sorokiniana*.

Significance
The pathogen produces ‘victorin’, a host-specific toxin which is especially destructive in oat varieties with the ‘Victoria’ parentage bred for crown rust resistance. Complete eradication of a susceptible crop is possible.

Symptoms
Infected plants show root rot, leaf blight, premature ripening and lodging in susceptible varieties of oat. Pathogenicity depends on the production and activity of the toxin ‘victorin’.

Hosts
Oat (Tveit 1956; Ivanoff 1959), wheat (Chidambaram *et al.* 1973) and a number of grasses listed by Luttrell (1951) and Meehan (1947).

Geographical distribution
Argentina, Australia, Bolivia, Brazil, Canada, India, Irish Republic, Malaysia, Saudi Arabia, Scotland, Switzerland, The Netherlands, USA, Zambia, Zimbabwe (CMI 1990).

Biology and transmission
Source of inoculum can be seeds carrying mycelium or dead plant tissue invaded with pseudothecia. Mycelium and conidia may remain viable in soil at 5°C for more than 10 years. Infection of the plant can take place in any stage of plant development.

Detection
Agar test or freezing blotter test.

Treatment
Seed treatment with a fungicide containing imazalil is recommended.

For further reading, see p. 63.

6. 'Septoria' diseases

6.1. Septoria avenae blotch, speckled blotch of oat

Cause
Teleomorph: *Leptosphaeria avenaria* f.sp. *avenaria* Weber, anamorph: *Septoria avenae* f.sp. *avenae* Frank. Size of the pycnidiospores sometimes leads to confusion with *S. nodorum* (Wiese 1987).

Significance
Occurs frequently and can cause reductions in yield; reported to be of increasing importance in Canada (Jones and Clifford 1983).

Symptoms
Purple-brown coloured leaf lesions/blotches with orange margins on leaf blades of mature plants. Pycnidia develop in the lesions. Lesions on leaves extend into the culm resulting in necrosis and blackening; similar lesions on floral bracts result in discolouration of kernels. Seed can become infected from infected glumes.

Hosts
Oat and other Gramineae (Dickson 1956; Seidel 1974).

Geographical distribution
Cosmopolitan.

Biology and transmission
The fungus is seedborne and can survive on stubble. Usually infected seedlings will not emerge. Other sources of primary inoculum are not important.

Detection
Microscopic investigation of the pycnidiospores.

Treatment
Seed treatment; no specific information available, see *Septoria nodorum*.

For further reading, see p. 63.

6.2. Septoria nodorum blotch, glume blotch

Cause
Teleomorph: *Leptosphaeria nodorum* E. Müller, anamorph: *Septoria nodorum* (Berk.) Berk.

Significance
S. nodorum, together with *S. tritici* and *S. avenae* f.sp. *triticea,* causes the 'septoria complex of wheat'. Worldwide yield losses of 2% annually are reported (Wiese 1987), but losses vary locally (King *et al.* 1983; Jones 1985). The increased importance of *S. nodorum in* some countries followed the intensification of cereal cropping (King *et al.* 1983). Losses are mainly due to grain shrivelling (Seidel 1974; Jones and Clifford 1983; Eyal *et al.* 1987).

Symptoms
Water-soaked lesions on all aerial parts of the plant, but more generally on the floral braces (Fig. 27) and nodal tissues of the culm. Lesions turn brown and necrotic; pycnidia (Fig. 28) develop in necrotic areas.

Hosts
S. nodorum has been isolated from hosts in 17 genera (Eyal *et al.* 1987). Besides wheat, the pathogen occurs on barley (Jones and Clifford 1983; King *et al.* 1983; Eyal *et al.* 1987), rye and other grasses (Seidel 1974), and on triticale (Abreu and Marques 1989). *Agropyron* and *Bromus* spp. are among the many alternative hosts (Krupinsky 1985, 1989).

Geographical distribution
Cosmopolitan (CAB IMI 1992), especially southeastern United States, South America, Europe, Africa and East Asia (Wiese 1987).

Biology and transmission
The teleomorph stage is reported from Australia, Chile, Ukraine and the USA (Eyal *et al.* 1987), as well as South Africa (Kemp *et al.* 1989). Mycelium and pycnidia in seed are the primary source of inoculum; the seedborne mycelium can cause seedling infection. Pseudothecia and pycnidia on wheat stubble are also infectious.

Fig. 27. Symptoms of glume blotch on a wheat spike. (Dr O.F. Mamluk, ICARDA, Aleppo)

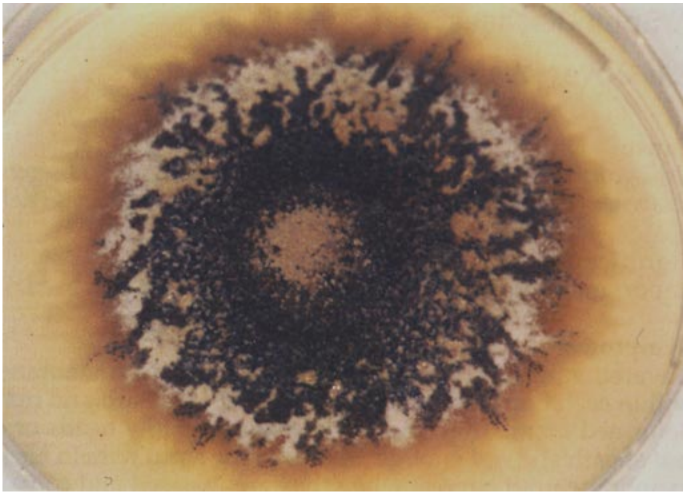

Fig. 28. Pycnidia of *Septoria nodorum* on agar medium. (Dr O.F. Mamluk, ICARDA, Aleppo)

Detection

Blotter test and microscopic examination of the pycnidiospores for distinction from other *Septoria* spp. through spore shape and size. *S. nodorum* develops readily pycnidia with pycnidiospores on media.

Treatment

Fungicide seed treatment: captafol (Jones and Clifford 1983; Obst 1989), prochloraz, propiconazole, carbendazim (Jones and Clifford 1983).

For further reading, see p. 63.

7. Smut diseases

7.1. Covered smut of barley

Cause
Ustilago hordei (Pers.) Lagerh. [syn. *Ustilago kolleri* Willie; *U. segetum* var. *hordei* (Pers.) Rabenh.; *U. avenae* (Pers.) Rostr. var. *levis* Kellerm and Sw.; *U. levis* (Kellerm. and Sw.) Magn.].

Significance
Important in areas where seed treatment is not practised.

Symptoms
Covered smut is characterized by a persistent membrane which encloses the smut within each floret. Smutted heads (Fig. 29) are hard and may contain parts of chaff and deformed areas. They emerge later than healthy heads or may be trapped in the flag leaf sheath and not emerge. Spores of the smut remain enclosed and compacted in the membrane until harvest when they are released and contaminate healthy grain.

Hosts
Oat, barley rye and various grasses in the genera *Agropyron* and *Elymus* (Fischer 1953; Neergaard 1979).

Geographical distribution
Cosmopolitan.

Biology and transmission
The smutted florets of infected heads remain intact until harvesting. Then the membrane breaks and teliospores are released which contaminate healthy seeds and the soil. Teliospores germinate at the same time as the seed. After successful infection the fungus becomes established behind the meristem. Infection occurs under high soil moisture conditions and between 14 and 25°C, with an optimum between 20 and 24°C. The fungus follows the growing point until flowering when it invades the ovary and replaces the seed with masses of teliospores.

Detection
Seed washing test.

Treatment
Seed treatment is effective but a lapse in its use can allow the disease to reappear quickly

For further reading, see p. 64.

Fig. 29. Covered smut of barley; healthy head on the right. (Dr D.E. Mathre, Montana State University, Bozeman, MT)

7.2. Flag smut

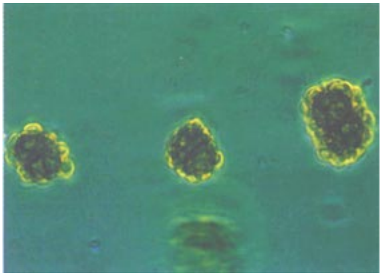

Fig. 30. Teliospores of *Urocystis agropyri*. (Dr M. Diekmann, IPGRI, Rome)

Cause
Urocystis agropyri (Preuss) Schröter [syn. *U. tritici* Körn.]. The spore balls consist of 1 to 4 dark teliospores (10 to 20 µm) (Fig. 30), surrounded by a layer of hyaline to brown sterile cells (total size 18 to 40 µm). *Urocystis occulta* Rabenh. is morphologically similar, with slightly larger spore balls (20 to 52 µm).

Significance
Unless controlled, causes significant yield losses in wheat, mainly in autumn-sown wheat in regions with arid summers and mild winters (Line 1993).

Symptoms
Infected plants show chlorotic striping parallel to the veins of leaf blades and sheaths which turn greyish-black (sori). The epidermis ruptures to release masses of dark-brown powdery teliospores. Leaves are twisted and split lengthwise (Fig. 31). Affected plants are dwarfed.

Hosts
For *U. agropyri* wheat, *Aegilops crassa, Lolium temulentum* and a number of other grasses (Mamluk *et al.* 1990, 1992). In Syria also reported on barley (Azmeh and Kousaji 1982). *U. occulta* reported only on rye.

Geographical distribution
Cosmopolitan.

Biology and transmission
Soilborne and externally seedborne. It can survive in dry soil for more than a year.

Detection
Centrifuge wash test.

Treatment
Preferably with systemic fungicides such as carboxin or triadimenol (Line 1972; Goel and Jhooty 1985).

For further reading, see p. 65.

Fig. 31. Flag smut of wheat. (Dr M. Diekmann, IPGRI, Rome)

7.3. Loose smut of wheat and barley

Cause
The disease is caused by two distinct pathogens: on wheat by *Ustilago tritici* (Pers.) Rostr. [syn. *Ustilago nuda* var. *tritici* Schaf.] and on barley by *Ustilago nuda* (Jens) Rostr.

Significance
Important in areas where seed treatment with appropriate systemic fungicides is not practised.

Symptoms
Loose smut appears after the ear has emerged. Some or all of the ears of infected plants apart from the rachis are replaced by a dark brown to olive-black powdery mass of spores (Fig. 32). Initially this mass of spores is covered by a membrane but this ruptures quickly to expose the smut. The spores are then easily blown away or washed off by rain so that as the crop matures, only the bare rachis with traces of spores and the membrane remain. In some cultivars, infected plants may have dark green erect leaves with chlorotic streaks which turn into necrotic streaks prior to the emergence of diseased ears.

Fig. 32. Loose smut of wheat. (Dr G.B. Wildermuth, Queensland Wheat Research Institute, Toowoomba, Qd)

Hosts

Barley for *U. nuda*, wheat, rye, triticale, *T. dicoccum* Schübler, *T. monococcum* L., *T. polonicum* L., *T. spelta* L., *T. turgidum* L.; various grasses in the genera *Aegilops, Agropyron, Elymus, Haynaldia* for *U. tritici* (Fischer 1953; Neergaard 1979; Wiese 1987).

Geographical distribution

Cosmopolitan.

Biology and transmission

Loose smut survives as dormant mycelium in the embryo of infected seed. When the seed germinates, the mycelium also grows and migrates towards the growing point of the plant. The fungus grows systemically in the host without causing symptoms. Spikelets are invaded intracellularly and all tissues except the rachis are converted to sori containing dry, dark brown teliospores. Initially these spores are enclosed in a membrane which quickly ruptures and teliospores are dispersed by wind and rain as far as 150 m. Spores are deposited on flowering spikes, germinate and promycelia fuse to produce infective hyphae. These hyphae penetrate the ovary wall and the fungus is reestablished within the developing grain. Infection occurs during wet, cloudy weather and cool to moderate temperatures (16 to 22°C). Within one week after flowering, the ovary becomes resistant to infection.

Detection

Embryo count method (Rennie 1982, 1988).

Treatment

Systemic seed treatments such as carboxin, carboxin plus thiram, carbendazim, benomyl, pyracarbolid, or terbutrazole (Agarwal *et al.* 1993).

For further reading, see p. 65.

7.4. Loose smut of oat

Cause
Ustilago segetum (Bull.) Rousel var. *avenae* (Pers.) Brun. [syn. *Ustilago avenae* (Pers.) Rostr.].

Significance
Not usually an important disease.

Symptoms
Loose smut occurs in the panicle where seed, hulls and glumes are replaced with a powdery mass of dark brown spores. Infected plants may be shorter than healthy plants. After the head emerges, spores are enclosed in a membrane which quickly ruptures allowing the spores to be blown away or washed off by rain. At maturity, only a few spores and fragments of the panicle remain.

Hosts
Oat, barley and various grasses in the genera *Arrhenatherum, Avena* and *Hordeum* (Fischer 1953; Neergaard 1979).

Geographical distribution
Cosmopolitan.

Biology and transmission
Loose smut of oat is carried over from season to season on the exterior of seeds, particularly between the caryopsis and the glumes. Spores germinate with the seedling, hyphae infect the seedling and follow the growing point. When the head emerges, spores are formed which are easily dispersed by wind to contaminate seeds.

Detection
Seed washing test. Spores of this fungus are brown and echinulate, 5 to 12 ìm in diameter. Minute echinulation of the outer surface of the spore distinguishes it from *Ustilago hordei* (Fischer 1953; Mordue and Ainsworth 1984).

Treatment
Loose smut of oat can be controlled effectively through seed treatment with a protective of systemic fungicide. The same treatment will also control covered smut on oat (Martens *et al.* 1984).

For further reading, see p. 65.

8. Wheat blast

Cause
Teleomorph: *Magnaporthe grisea* (Hebert), anamorph: *Pyricularia grisae* (Cooke) Sacc. The fungus is morphologically indistinguishable from *P. oryzae* Cav. The latter name is usually retained for the forms on rice.

Significance
The disease has become a limiting factor affecting wheat production in Brazil (Urashima *et al.* 1993).

Symptoms
Most damage results from infection of the rachis causing partial or total sterility of spikes. Frequently, all the spikelets above the infection point turn white (Fig. 33). However, all foliar parts may be infected. Lesions on leaves, culms and glumes resemble those of ‘rice blast’ and are elliptical to elongated with white to light brown centres and dark gray to reddish-brown borders. Sporulation occurs on the underside of leaves. Disease on seedlings can be severe although the incidence of foliar lesions generally decreases as the crop ages. Seeds infected during early stages of development are severely shriveled and usually killed. However, those infected later may appear healthy and provide a primary source of inoculum.

Hosts

- According to Urashima *et al.* (1993) isolates from wheat did not infect rice, nor grasses such as *Digitaria* spp. or *Brachiaria* spp. The same isolates produced symptoms on barley, maize; oat, rye, and sorghum as well as *Dactylis glomerata, colium* spp., *Festuca* spp. and others. Conversely, some isolates from rice were capable of infecting wheat.

Fig. 33. White spikes caused by wheat blast. (Dr G. Hettel, CIMMYT, El Batan)

Geographical distribution

On wheat reported from India (McRae 1922), Pakistan (Malik and Khan 1943), USA (Rush and Carver 1973) and Brazil (Igarashi *et al.* 1986).

Biology and transmission

The pathogen is seed-transmitted (Menten and Morais 1987). Wind- and rain-borne conidia from lesions on the undersurfaces of infected leaves spread the disease within the crop. Disease development is promoted by high moisture and temperatures above 22°C (Igarashi 1988). Although *P. oryzae* on rice is found wherever the crop is grown throughout the world, and has a very wide host range, wheat blast appears to be very limited by environment.

Detection

Freezing blotter test.

Treatment

Seed treatments effective against *P. oryzae* on rice are presumed to have similar activity against the pathogen in wheat seed. Thiram + benomyl has been successfully used in Japan (Nakamura 1986) on rice. Iprodione + thiram has been reported as very effective on wheat (Igarashi 1990).

For further reading, see p. 66.

Nematodes

1. Wheat gall nematode

Cause
Anguina tritici (Steinbuch) Chitwood [syn. *Vibrio tritici* Steinbuch; *Anguillula tritici* (Steinbuch) Grube; *Anguillulina tritici* (Steinbuch) Gervais and Beneden; *Tylenchus tritici* (Steinbuch) Bastian; *Anguillula scandens* Schneider].

Significance
Important in areas where modern seed cleaning methods and proper crop rotation are not practised. Losses up to 30% were reported in Iraq (Stephan 1988).

Symptoms
Infected seedlings are stunted and show characteristic rolling, twisting and crinkling of leaves, resulting in distorted plants (Fig. 34). At the end of the season small brown to black galls are found in the ears of plants (Fig. 35). May be confused with bunted kernels, but unlike these the nematode galls are hard and may not be crushed easily.

Hosts
Wheat and other *Triticum* spp., rye (Goodey *et al.* 1965). Oat and barley were reported hosts but with little or no reproduction (Southey 1972).

Geographical distribution
Cosmopolitan

Fig. 34. Distorted wheat plants infected by *Anguina tritici.* (Dr W. Abu-Gharbieh, University of Jordan, Amman)

Fig. 35. Galls of *Anguina tritici* mixed with healthy wheat seeds. (Society of Nematologists)

Biology and transmission

Galls already in the soil or sown with contaminated seeds are the sources of infestation. In the field, galls become moist and soft, facilitating release of second stage juveniles (J2). Juveniles move progressively on the growing tip until they penetrate the floral primordia, where they develop to maturity. Females lay thousands of eggs in the seed gall; eggs hatch and J2 enter anhydrobiosis. There is one generation per year.

Detection

Visual inspection for black galls. Seed testing is performed by overnight soaking of galls in water. When the swollen gall is pierced with a needle, thousands of juveniles burst from the gall.

Treatment

Most effective control is by mechanical seed cleaning. Galls may be also removed by flotation in 20% brine solution, followed by thorough washing in water. Hot water treatment of 54°C for 10 min is also reported to be effective (Swarup *et al*, 1993).

For further reading, see p. 66.

2. Summer dwarf nematode

Cause
Aphelenchoides fragariae (Ritzema Bos) Christie [syn. *Aphelenchus fragariae* Ritzema Bos; *Aphelenchus olesistus* Ritzema Bos; *Aphelenchoides olesistus* Ritzema Bos and Steiner; *Aphelenchus pseudolesistus* Goodey; *Aphelenchoides pseudolesistus* Goodey].

Significance
Most important on strawberries.

Symptoms
Leaf blotches on ferns occur in stripes, and on flowering plants as irregular water-soaked patches later turning brown, violet or purple. The foliage of various host plants may exhibit die-back, dwarfing, decay of buds and blackened flower heads. On strawberries, the nematode causes the 'spring dwarf' disease, along with malformation and discolouration of leaves (Siddiqi 1975).

Hosts
Over 250 plants in 47 families are recorded as hosts from temperate and tropical zones, including strawberry, ferns, common weeds, aquatic plants, flowering plants (Goodey *et al.* 1965; Siddiqi 1975).

Geographical distribution
A. fragariae is a common and widespread species mainly encountered in moist conditions. It was reported from Denmark, Hawaii, Germany, India, Ireland, Italy Japan, Poland, Sweden, UK, the USA and the former USSR (Siddiqi 1975).

Biology and transmission
A. fragariae is an obligate parasite of aboveground plant parts and may be endo- or ectoparasitic. The life cycle is considered short and is completed within 10 to 12 days at 20°C. The nematode may overwinter in basal buds of perennials or enter quiescence in infected dry leaves. Dissemination occurs by infected plant parts, runners, cuttings, or seeds.

Detection
The nematode can be recovered from plant materials or soil using standard extraction methods (Ayoub 1977).

Treatment
Hot water treatment of dormant stocks. On young plants, foliar spraying and/or soil drenching with nematicides (e.g. oxamyl, aldicarb, fenamiphos) can be used (Siddiqi 1975).

For further reading, see p. 67.

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